TRPA1

- Conditioned medium from painful non-NF2 schwannomatosis tumors increases pain behaviors in mice
- Sex differences in the vasoactive effect of transient receptor potential channels: TRPM3 as a new therapeutic target for (neuro)vascular disorders
- Sciatic nerve stimulation alleviates neuropathic pain and associated neuroinflammation in the dorsal root ganglia in a rodent model
- Remote neurostimulation through an endogenous ion channel using a near-infrared light-activatable nanoagonist
- Inflammation-induced mast cell-derived nerve growth factor: a key player in chronic vulvar pain?
- The pathophysiology of trigeminal neuralgia: a molecular review
- Micromotion Derived Fluid Shear Stress Mediates Peri-Electrode Gliosis through Mechanosensitive Ion Channels
- Modification of the TRP Channel TRPA1 as a Relevant Factor in Migraine-Related Intracranial Hypersensitivity

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Astrocytic transient receptor potential ankyrin 1 (TRPA1) in glial phenotype transformation in neuroinflammation after intracerebral hemorrhage (ICH). Wild-type astrocytes and TRPA1-/- astrocytes were subjected to 6-h hemin treatment, and the calcium ions and transcriptome sequencing were assessed. A mouse autologous blood injection ICH model was established to evaluate the proliferation and phenotypes of astrocytes and microglia around the hematoma. The neuroinflammation and behavioral performance of wild-type ICH mice and TRPA1-/- ICH mice were assessed. Knockout of astrocytic TRPA1 decreased calcium ions of astrocytes after hemin treatment in-vitro, and microglial and astrocytes around the hematoma proliferated after the ICH model. Furthermore, RNA-sequencing (RNA-seq), immunofluorescence, and Western blotting results showed that the activated astrocytes transformed into the A2 phenotype in TRPA1-/- ICH mice. The 'ameboid' microglia were observed around the hematoma in TRPA1-/- ICH mice. The proliferation of A2 astrocytes and 'ameboid' microglia ameliorated the neuroinflammation after ICH. The inflammatory response was reduced by inhibiting the mitogen-activated protein kinase/nuclear factor kappa-B signaling pathway, and neurologic deficits were improved in TRPA1-/- ICH mice compared with wild-type ICH mice. This research suggests that astrocytic TRPA1 is a new therapeutic target to rescue neuroinflammation by modulating the glial phenotype after ICH [1)]

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Xia M, Chen YJ, Chen B, Ru X, Wang J, Lin J, Tang X, Chen W, Hu R, Li W, Feng H. Knockout of transient receptor potential ankyrin 1 (TRPA1) modulates the glial phenotype and alleviates perihematomal neuroinflammation after intracerebral hemorrhage in mice via MAPK/NF-κB signaling. Neuroreport. 2023 Feb 1;34(2):81-92. doi: 10.1097/WNR.0000000000001862. Epub 2022 Nov 30. PMID: 36608163.